functional recovery (EF pre: 49 ± 9%, post: 58 ± 12%, $p < 0.05$) and a lower degree of replacement fibrosis, compared to pts with INT > 30 days (n = 16; EF pre: 40 ± 11%, post: 45 ± 11%, $p < 0.05$).

Hibernating myocardium seems to represent a rather unstable condition, in which by time a progressive structural degeneration might appear. For a better prognosis, early revascularization is mandatory to salvage chronically jeopardized but viable myocardium with improvement of functional outcome.

1015-129 Cardiac Pseudoaneurysm: Clinical Profile and Outcome in 55 Cases

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Although cardiac pseudoaneurysm (PA) is potentially fatal, there is limited knowledge of its clinical profile and natural history. Accordingly, we reviewed the clinical profile and outcome of 55 patients (67% males, median age = 58) with PA who were seen at the Mayo Clinic between 1980 and 1996. Etiology of PA was post cardiac surgery in 30 (54.5%), post myocardial infarction in 22 (40%), and endocarditis in 3 (5.5%). Prior surgery for complex congenital heart disease was the cause in 15 (27.3%) patients. Diagnostic methods were two-dimensional Doppler echocardiography in 40, cardiac catheterization in 35, MRI in 5 and CT scan in 4. CXR showed a localised bulge in 5 (9.1%) patients. PA was discovered incidentally in 23 (41.8%) patients who were asymptomic. Five (9.1%) patients presented acutely, 3 as acute myocardial infarction and 2 in cardiac tamponade. Other clinical presentations included congestive heart failure in 9 (16.4%), chest pain in 7 (12.7%), syncope/arrhythmia in 5 (9.1%) and systemic embolism in 3 (5.5%). Typical locations of PA were: post myocardial infarction – inferior/posterior lateral (18, 81.8%); post congenital heart surgery – right ventricular outflow tract (13, 86.7%); post mitral valve replacement – posterior subannular (4, 100%) and post aortic valve replacement – subaortic (3, 100%). Forty four (80%) patients underwent surgical repair with an operative mortality of 6.8%. Ten (18.2%) patients did not have surgery. Six of these patients died after a median duration of 2.1 years (11 days to 4.7 years), 3 of non-cardiac causes (median survival = 3.2 years, range = 0.9 to 4.7 years). Survival at 2 years was 63%.

Conclusions: 1) Most PA occured after myocardial infarction or cardiac surgery. 2) A significant number of patients were asymptomatic. 3) Repair of PA was associated with a low operative mortality. 4) Contrary to previous beliefs, outcome was not dismal in patients not treated surgically and immediate surgery may not be mandatory for these patients.

1015-130 Increased Levels of Cardiac Troponin I in Cancer Patients

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Early-stage cancer patients are often treated with multidrug cytoreductive chemotherapy that includes doxorubicin. The usefulness of the drug is limited by a dose-dependent cardiac toxicity. Cardiac troponin I (cTnI) is a structural intracellular regulatory protein and a highly specific marker for myocardial injury. We measured serum cTnI in 25 consecutive patients with adenocarcinomas (small cell lung, kidney, prostate and bladder cancer) and in 30 control subjects with a newly developed highly sensitive immunoenzymometric assay (detection limit, 3 pg/mL). Eight patients had been given an intermediate cumulative dose of doxorubicin (250–300 mg/m^2) when included in the study, while the remaining 17 patients were assessed before chemotherapy. Doxorubicin-treated patients demonstrated significantly higher cTnI levels (mean, 70.5 ± 15.7, 95% confidence interval, 57.4–83.6 pg/mL) compared to control subjects (mean, 10.6 ± 15.7, 95% confidence interval, 4.7–16.5 pg/mL, $p = 0.0001$) and untreated patients (51.3 ± 22.0, 95% confidence interval, 40.0–62.7 pg/mL, $p = 0.007$), respectively. Left ventricular ejection fraction was significantly reduced in doxorubicin-treated patients (0.42 ± 0.12) and within the normal range in untreated patients (0.61 ± 0.1, $p = 0.0004$). This study is the first to show that cTnI, a sensitive marker for myocardial sufferance, is elevated in cancer patients, with a further increase in patients given doxorubicin. These data raise new intriguing hypotheses, specifically, whether the presence of neoplasia has effects on cardiomyocytes, and whether cardiac toxicity of antineoplastic drugs could be monitored with cTnI.

1015-131 Abnormalities in Left Ventricular Shape Occur Despite Normal Ejection Fraction in the Hearts of Patients With Systemic Sclerosis

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Cardiac dysfunction in systemic sclerosis (SS) is a late sign and of bad prognosis. We wondered whether changes in regional and global LV geometry could be present prior to LV volume and functional changes. To explore this, we performed quantitative shape analysis of the LV in 2 chamber (2C) and long axis (LAX) views of 19 pts with SS and compared them to 35 matched normals (N). Digitized diastolic (D) and systolic (S) LV endocardial outlines were analyzed to get an index of global shape (Fourier Power Index or PI; lower PI = rounder, F3, third component = elongation) and of regional shape (quantitative Curvature or C). *Results:* (* $p < 0.05$ compared to N) (Means ± SE) ED volumes were comparable in both groups but ES volumes were bigger in SS (N: 18 ± 9 ml vs SS: 40 ± 22 ml). Although % LVEF was lower in SS than in N (N: 77% ± 9 vs SS: 60% ± 13), the EF was still in the normal range. *Shape analysis indicated a number of heterogenous geometric changes in SS pts.* They were: In diastole, no global change in shape (PI) was detected in LAX but in systole, there was more elongation (*F3 in N: 0.47 ± 0.03, SS: 0.55 ± 0.02**). In the 2C view comprising of anterior and inferior walls, decreased roundness in diastole was noted. (*PI in N: 15 ± 1.14, SS: 23 ± 2.8**). Also, dynamic shape change during the cardiac cycle (% ΔPI) was blunted (*N: 156% ± 15 vs SS: 74% ± 20**). C analysis showed a more curved posterior wall in diastole (C in *N: 6.1 ± 0.4 vs SS: 7.8 ± 0.7**), a flatter anterior septum in systole (*N: 7.3 ± 1 vs SS: 4.3 ± 0.7**), and diminution of normal concavity of the inferior wall from diastole to systole (*N C: −5 ± 0.5 to −3.2 ± 0.6 vs SS C: −1.3 ± 0.7 to −1 ± 1.1**). The S Apical C was higher in SS (*N: 28 ± 1.8 vs SS: 37 ± 2.7**) *Conclusion:* Heterogeneous geometric changes occur in systemic sclerosis pts, possibly related to patchy myocardial fibrosis. LV shape abnormalities are potential markers of subclinical cardiac involvement in this disorder.

1015-132 Regional Wall Motion Abnormalities in Diabetic Hearts Without Coronary Artery Disease Closely Relate Microvascular Circulation Disturbance

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Disorders of myocardial energy metabolism, sympathetic nerve disfunction, and microvascular disease are the possible causes of the regional wall motion abnormalities (rWMA) in NIDDM patients without coronary artery disease (CAD). To assess the causes of the rWMA, we estimated the relationships among rWMA indicated by left ventriculography (LVG), regional myocardial fatty acid metabolism evaluated by iodine-123 labeled beta-methyl-iodophenylpentadecanoic acid myocardial SPECT (BMIPP), sympathetic nerve function estimated by iodine-123 labeled metaiodobenzylguanidine myocardial SPECT (MIBG), and coronary small vessel abnormality indicated by dipyridamole thallium-201 myocardial SPECT (Dip). Subjects were twenty four NIDDM patients (age: 61 ± 10 y, M/F: 13/11) who have undergone coronary angiography and did not have CAD. For regional analysis, we divided LV into five regions (anterior, septal, apex, lateral, infero-posterior) in LVG, BMIPP, MIBG and Dip, and assessed the coincidence with rWMA, regional BMIPP uptake, regional MIBG uptake and redistribution phenomenon (RD) in Dip. 9 of all subjects (22 of 120 regions) had rWMA. BMIPP uptake in the 48 regions of all were reduced. All defects of MIBG SPECT were showed in inferior wall, and did not indicate any regionality. RD was seen in 24 regions. Sensitivity of RD for detecting the rWMA was 45% and similarly that of defects in BMIPP was 59%. However, specificity of RD (82%) was significantly higher than the defects in BMIPP (52%). Thus, these results suggest that the rWMA in NIDDM may relate to small vessel abnormality more closely than the disorder of regional myocardial fatty acid metabolism.

1015-133 Effect of Left Atrial Appendage on Left Ventricular Filling Dynamics: A Computational Model

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The effect of the hemodynamic characteristics of the left atrial appendage (LAA) on the atrioventricular filling system are difficult to study in vivo. A computational model of the canine pulmonary circulation, left atrium (LA) and ventricle (LV) was modified to simulate the effects of LAA compliance and contractility on pulmonary venous, LAA and mitral flow; LA and LV pressure and volume; and cardiac output. LAA flow patterns are consistent with those obtained by transesophageal Doppler: rapid LAA emptying into